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 NEWS  7  DEC 21   IPC search and display fields enhanced in CA/CAplus with the
                   IPC reform
 NEWS  8  DEC 23   New IPC8 SEARCH, DISPLAY, and SELECT fields in USPATFULL/
                   USPAT2
 NEWS  9  JAN 13   IPC 8 searching in IFIPAT, IFIUDB, and IFICDB
 NEWS 10  JAN 13   New IPC 8 SEARCH, DISPLAY, and SELECT enhancements added to
                   INPADOC
 NEWS 11  JAN 17   Pre-1988 INPI data added to MARPAT
 NEWS 12  JAN 17   IPC 8 in the WPI family of databases including WPIFV
 NEWS 13  JAN 30   Saved answer limit increased
 NEWS 14  JAN 31   Monthly current-awareness alert (SDI) frequency
                   added to TULSA

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               CURRENT MACINTOSH VERSION IS V6.0c(ENG) AND V6.0Jc(JP),
               AND CURRENT DISCOVER FILE IS DATED 19 DECEMBER 2005.
               V8.0 USERS CAN OBTAIN THE UPGRADE TO V8.01 AT
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FILE COVERS 1907 - 14 Feb 2006 VOL 144 ISS 8
FILE LAST UPDATED: 13 Feb 2006 (20060213/ED)


```
=> "adenovirus vector"
         23546 "ADENOVIRUS"
          3444 "ADENOVIRUSES"
         24188 "ADENOVIRUS"
                 ("ADENOVIRUS" OR "ADENOVIRUSES")
        152523 "VECTOR"
         94931 "VECTORS"
        207686 "VECTOR"
                 ("VECTOR" OR "VECTORS")
L1        4224 "ADENOVIRUS VECTOR"
                 ("ADENOVIRUS"(W)"VECTOR")

=> Hela
         31317 HELA
            23 HELAS
L2       31337 HELA
                 (HELA OR HELAS)

=> L1 and L2
L3         135 L1 AND L2

=> S3 and L3
         11477 S3
L4           3 S3 AND L3

=> D L4 IBIB ABS 1-3

L4   ANSWER 1 OF 3  CAPLUS  COPYRIGHT 2006 ACS on STN
ACCESSION NUMBER:        2005:1328672  CAPLUS
DOCUMENT NUMBER:         144:65123
TITLE:                   Construction of recombinant adenovirus
                         vector specifically expressing immune
                         modulatory factor GM-CSF in tumor cells and uses
                         thereof
INVENTOR(S):             Ke, Zunhong
PATENT ASSIGNEE(S):      Chengdu Kanghong Technology Enterprises (Group) Co.,
                         Ltd., Peop. Rep. China
SOURCE:                  PCT Int. Appl., 78 pp.
                         CODEN: PIXXD2
DOCUMENT TYPE:           Patent
LANGUAGE:                Chinese
FAMILY ACC. NUM. COUNT:  1
PATENT INFORMATION:

     PATENT NO.           KIND   DATE        APPLICATION NO.         DATE
     ---------------      ----   --------    --------------------    --------
     WO 2005121343         A1    20051222    WO 2004-CN1321          20041119
         W:  AE, AG, AL, AM, AT, AU, AZ, BA, BB, BG, BR, BW, BY, BZ, CA, CH,
             CN, CO, CR, CU, CZ, DE, DK, DM, DZ, EC, EE, EG, ES, FI, GB, GD,
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GE, GH, GM, HR, HU, ID, IL, IN, IS, JP, KE, KG, KP, KR, KZ, LC,
LK, LR, LS, LT, LU, LV, MA, MD, MG, MK, MN, MW, MX, MZ, NA, NI,
NO, NZ, OM, PG, PH, PL, PT, RO, RU, SC, SD, SE, SG, SK, SL, SY,
TJ, TM, TN, TR, TT, TZ, UA, UG, US, UZ, VC, VN, YU, ZA, ZM, ZW
RW: BW, GH, GM, KE, LS, MW, MZ, NA, SD, SL, SZ, TZ, UG, ZM, ZW, AM,
AZ, BY, KG, KZ, MD, RU, TJ, TM, AT, BE, BG, CH, CY, CZ, DE, DK,
EE, ES, FI, FR, GB, GR, HU, IE, IS, IT, LU, MC, NL, PL, PT, RO,
SE, SI, SK, TR, BF, BJ, CF, CG, CI, CM, GA, GN, GQ, GW, ML, MR,
NE, SN, TD, TG

PRIORITY APPLN. INFO.: CN 2004-10046237 A 20040607

AB The present invention relates to construction of recombinant **adenovirus vector** specifically expressing immune modulatory factor GM-CSF in tumor cells and its uses thereof. Specifically, the invention relates to gene therapy for tumors, specifically, it relates to the construction of oncolytic recombinant **adenovirus vector**, which specifically expresses immune modulatory cytokines, including IL-2, IL-10, IL-12, IL-15, IL-24, GM-CSF, G-CSF, INF-α and INF-β, in tumor cells and the methods for construction and therapeutic uses thereof.

REFERENCE COUNT: 7 THERE ARE 7 CITED REFERENCES AVAILABLE FOR THIS RECORD. ALL CITATIONS AVAILABLE IN THE RE FORMAT

L4 ANSWER 2 OF 3 CAPLUS COPYRIGHT 2006 ACS on STN

ACCESSION NUMBER: 2004:899614 CAPLUS
TITLE: Method for production of oncolytic adenoviruses
INVENTOR(S): Kadan, Michael; Kaptur, Ronald; Brousseau, David; Mittelstaedt, Denise; Li, Yuanhao
PATENT ASSIGNEE(S): Novartis Ag, Switz.
SOURCE: PCT Int. Appl.
CODEN: PIXXD2
DOCUMENT TYPE: Patent
LANGUAGE: English
FAMILY ACC. NUM. COUNT: 1
PATENT INFORMATION:

PATENT NO.	KIND	DATE	APPLICATION NO.	DATE
WO 2004092348	A2	20041028	WO 2004-US11855	20040415
WO 2004092348	A3	20050310		

W: AE, AG, AL, AM, AT, AU, AZ, BA, BB, BG, BR, BW, BY, BZ, CA, CH, CN, CO, CR, CU, CZ, DE, DK, DM, DZ, EC, EE, EG, ES, FI, GB, GD, GE, GH, GM, HR, HU, ID, IL, IN, IS, JP, KE, KG, KP, KR, KZ, LC, LK, LR, LS, LT, LU, LV, MA, MD, MG, MK, MN, MW, MX, MZ, NA, NI, NO, NZ, OM, PG, PH, PL, PT, RO, RU, SC, SD, SE, SG, SK, SL, SY, TJ, TM, TN, TR, TT, TZ, UA, UG, US, UZ, VC, VN, YU, ZA, ZM, ZW
RW: BW, GH, GM, KE, LS, MW, MZ, SD, SL, SZ, TZ, UG, ZM, ZW, AM, AZ, BY, KG, KZ, MD, RU, TJ, TM, AT, BE, BG, CH, CY, CZ, DE, DK, EE, ES, FI, FR, GB, GR, HU, IE, IT, LU, MC, NL, PL, PT, RO, SE, SI, SK, TR, BF, BJ, CF, CG, CI, CM, GA, GN, GQ, GW, ML, MR, NE, SN, TD, TG

US 2005095705	A1	20050505	US 2004-824796	20040414

PRIORITY APPLN. INFO.: US 2003-463143P P 20030415

AB **HeLa-S3** cells comprising replication-competent **adenovirus vectors** are provided. Also provided are **HeLa-S3** producer cell lines and methods for producing replication-competent adenovirus using the same.

L4 ANSWER 3 OF 3 CAPLUS COPYRIGHT 2006 ACS on STN

ACCESSION NUMBER: 1998:268518 CAPLUS
DOCUMENT NUMBER: 128:318022
TITLE: DNA sequences encoding fusions of DNA repair proteins and their uses for inhibition action of chemotherapy agents in non-target cells
INVENTOR(S): Kelley, Mark; Williams, David
PATENT ASSIGNEE(S): Advanced Research & Technology Institute, USA
SOURCE: PCT Int. Appl., 150 pp.
CODEN: PIXXD2
DOCUMENT TYPE: Patent
LANGUAGE: English

FAMILY ACC. NUM. COUNT: 1
PATENT INFORMATION:

PATENT NO.	KIND	DATE	APPLICATION NO.	DATE
WO 9817684	A2	19980430	WO 1997-US19629	19971024
W: AL, AM, AT, AU, AZ, BA, BB, BG, BR, BY, CA, CH, CN, CU, CZ, DE, DK, EE, ES, FI, GB, GE, GH, HU, ID, IL, IS, JP, KE, KG, KP, KR, KZ, LC, LK, LR, LS, LT, LU, LV, MD, MG, MK, MN, MW, MX, NO, NZ, PL, PT, RO, RU, SD, SE, SG, SI, SK, SL, TJ, TM, TR, TT, UA, UG, US, UZ, VN, YU, ZW, AM, AZ, BY, KG, KZ, MD, RU, TJ, TM				
RW: GH, KE, LS, MW, SD, SZ, UG, ZW, AT, BE, CH, DE, DK, ES, FI, FR, GB, GR, IE, IT, LU, MC, NL, PT, SE, BF, BJ, CF, CG, CI, CM, GA, GN, ML, MR, NE, SN, TD, TG				
AU 9851953	A1	19980515	AU 1998-51953	19971024
US 6046036	A	20000404	US 1997-957302	19971024
US 6252048	B1	20010626	US 2000-542403	20000403
PRIORITY APPLN. INFO.:			US 1996-29308P	P 19961025
			US 1997-957302	A1 19971024
			WO 1997-US19629	W 19971024

AB Described are DNA-repair fusion proteins of multiple DNA repair proteins possessing the activity of each protein, and their related polynucleotide sequences and vectors. Thus, chimeric proteins were generated from human O-6-methylguanine-DNA methyltransferase (MGMT) and human apurinic/apyrimidinic endonuclease (APE). The chimera included fusions of full-length proteins and with N-terminally truncated APE. Overlapping PCR techniques were used to construct expression vectors in which the cDNAs are expressed by the murine phosphoglycerate kinase promoter and containing the SV40 small T intron and poly(A) tract. The proteins, when expressed in cells, e.g., hematopoietic cells, increase the survival rate of the cells when contacted with chemotherapeutic agents. In mammalian **HeLa** cells, chimerics expressing MGMT-APE have a 2-fold survival enhancement over untreated **HeLa** cells at a BCNU [1,3-bis(2-chloroethyl)-1-nitrosourea] of 75 μM, whereas at 150 μM BCNU there is a 4-8 fold enhancement. Protection was also observed against MMS (Me methanesulfonate). Also described are transgenic animal models wherein these proteins are expressed in essentially all cells of the animal. Such animal models are useful for instance in testing chemotherapeutic agents.

```
=> adenovirus
         23546 ADENOVIRUS
          3444 ADENOVIRUSES
L5       24188 ADENOVIRUS
                (ADENOVIRUS OR ADENOVIRUSES)

=> L5 and l2
L6        1706 L5 AND L2

=> S3 and L6
         11477 S3
L7          15 S3 AND L6

=> D L7 IBIB ABS 1-7

L7   ANSWER 1 OF 15  CAPLUS  COPYRIGHT 2006 ACS on STN
ACCESSION NUMBER:        2005:1328672  CAPLUS
DOCUMENT NUMBER:         144:65123
TITLE:                   Construction of recombinant adenovirus
                         vector specifically expressing immune modulatory
                         factor GM-CSF in tumor cells and uses thereof
INVENTOR(S):             Ke, Zunhong
PATENT ASSIGNEE(S):      Chengdu Kanghong Technology Enterprises (Group) Co.,
                         Ltd., Peop. Rep. China
SOURCE:                  PCT Int. Appl., 78 pp.
                         CODEN: PIXXD2
DOCUMENT TYPE:           Patent
LANGUAGE:                Chinese
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FAMILY ACC. NUM. COUNT: 1
PATENT INFORMATION:

PATENT NO.	KIND	DATE	APPLICATION NO.	DATE
WO 2005121343	A1	20051222	WO 2004-CN1321	20041119

W: AE, AG, AL, AM, AT, AU, AZ, BA, BB, BG, BR, BW, BY, BZ, CA, CH, CN, CO, CR, CU, CZ, DE, DK, DM, DZ, EC, EE, EG, ES, FI, GB, GD, GE, GH, GM, HR, HU, ID, IL, IN, IS, JP, KE, KG, KP, KR, KZ, LC, LK, LR, LS, LT, LU, LV, MA, MD, MG, MK, MN, MW, MX, MZ, NA, NI, NO, NZ, OM, PG, PH, PL, PT, RO, RU, SC, SD, SE, SG, SK, SL, SY, TJ, TM, TN, TR, TT, TZ, UA, UG, US, UZ, VC, VN, YU, ZA, ZM, ZW

RW: BW, GH, GM, KE, LS, MW, MZ, NA, SD, SL, SZ, TZ, UG, ZM, ZW, AM, AZ, BY, KG, KZ, MD, RU, TJ, TM, AT, BE, BG, CH, CY, CZ, DE, DK, EE, ES, FI, FR, GB, GR, HU, IE, IS, IT, LU, MC, NL, PL, PT, RO, SE, SI, SK, TR, BF, BJ, CF, CG, CI, CM, GA, GN, GQ, GW, ML, MR, NE, SN, TD, TG

PRIORITY APPLN. INFO.: CN 2004-10046237 A 20040607

AB The present invention relates to construction of recombinant **adenovirus** vector specifically expressing immune modulatory factor GM-CSF in tumor cells and its uses thereof. Specifically, the invention relates to gene therapy for tumors, specifically, it relates to the construction of oncolytic recombinant **adenovirus** vector, which specifically expresses immune modulatory cytokines, including IL-2, IL-10, IL-12, IL-15, IL-24, GM-CSF, G-CSF, INF-α and INF-β, in tumor cells and the methods for construction and therapeutic uses thereof.

REFERENCE COUNT: 7 THERE ARE 7 CITED REFERENCES AVAILABLE FOR THIS RECORD. ALL CITATIONS AVAILABLE IN THE RE FORMAT

L7 ANSWER 2 OF 15 CAPLUS COPYRIGHT 2006 ACS on STN
ACCESSION NUMBER: 2005:248643 CAPLUS
DOCUMENT NUMBER: 142:274056
TITLE: Sequences of human schizophrenia related genes and use for diagnosis, prognosis and therapy
INVENTOR(S): Liew, Choong-Chin
PATENT ASSIGNEE(S): Chondrogene Limited, Can.
SOURCE: U.S. Pat. Appl. Publ., 156 pp., Cont.-in-part of U.S. Ser. No. 802,875.
CODEN: USXXCO
DOCUMENT TYPE: Patent
LANGUAGE: English
FAMILY ACC. NUM. COUNT: 47
PATENT INFORMATION:

PATENT NO.	KIND	DATE	APPLICATION NO.	DATE
US 2004241727	A1	20041202	US 2004-812731	20040330
US 2004014059	A1	20040122	US 2002-268730	20021009
US 2005191637	A1	20050901	US 2004-803737	20040318
US 2005196762	A1	20050908	US 2004-803759	20040318
US 2005196763	A1	20050908	US 2004-803857	20040318
US 2005196764	A1	20050908	US 2004-803858	20040318
US 2005208505	A1	20050922	US 2004-803648	20040318
US 2004241727	A1	20041202	US 2004-812731	20040330

PRIORITY APPLN. INFO.:
US 1999-115125P P 19990106
US 2000-477148 B1 20000104
US 2002-268730 A2 20021009
US 2003-601518 A2 20030620
US 2004-802875 A2 20040312
US 2004-812731 A 20040330

AB The present invention is directed to detection and measurement of gene transcripts and their equivalent nucleic acid products in blood. Specifically provided is anal. performed on a drop of blood for detecting, diagnosing and monitoring diseases using gene-specific and/or tissue-specific primers. The present invention also describes methods by which delineation of the sequence and/or quantitation of the expression levels of disease-specific genes allows for an immediate and accurate diagnostic/prognostic test for disease or to assess the effect of a particular treatment regimen. [This abstract record is one of 3 records for

this document necessitated by the large number of index entries required to fully index the document and publication system constraints.].

L7 ANSWER 3 OF 15 CAPLUS COPYRIGHT 2006 ACS on STN
ACCESSION NUMBER: 2005:244695 CAPLUS
DOCUMENT NUMBER: 143:58180
TITLE: Safety characterization of **HeLa**-based cell substrates used in the manufacture of a recombinant adeno-associated virus-HIV vaccine
AUTHOR(S): Tatalick, Lauren M.; Gerard, Christopher J.; Takeya, Ryan; Price, David N.; Thorne, Barbara A.; Wyatt, Lisa M.; Anklesaria, Pervin
CORPORATE SOURCE: Targeted Genetics Corporation, Seattle, WA, 98101, USA
SOURCE: Vaccine (2005), 23(20), 2628-2638
CODEN: VACCDE; ISSN: 0264-410X
PUBLISHER: Elsevier B.V.
DOCUMENT TYPE: Journal
LANGUAGE: English
AB The use of transformed cell substrates for prophylactic vaccine manufacturing is widely debated. Extensive characterization is required to address the suitability of neoplastic cell substrates for vaccine manufacture The **HeLa**-based cell substrate used in the manufacture of a prophylactic rAAV-HIV vaccine, AAV2-gagPRΔRT (tgAAC09) was tested in vivo for its tumor-forming potential, the oncogenic potential of its high mol. weight DNA and the potential presence of occult oncogenic adventitious agents. This data from these in vivo studies, in conjunction with prion gene and protein characterization, cell and viral clearance studies and quantity of residual host-cell DNA levels in the purified tgAAC09 vaccine, were used to establish what the authors believe to be an acceptable safety profile for the vaccine manufacturing process. The tumor-producing dose in 50% of the animals was consistent with that in a published report from FDA staff for **HeLa** cells. High mol. weight cellular DNA was not oncogenic and no occult oncogenic agents were detected by testing in nude mice and newborn rodent models, resp. Endogenous prion protein was also normal and genomic sequence anal. detected no mutations associated with increased risk of prion disease. In addition, the purification process used to produce this vaccine candidate removed all detectable cells (clearance of greater than 22 log10), viral clearance study showed 6-17 log10 clearance of three model viruses and host-cell DNA in the bulk product was less than 100 pg host-cell DNA per dose of 3+1011 DNase resistant particles (DRP) of the vaccine. Taken together, the data from the in vivo and in vitro tests that were performed to characterize the **HeLa** based producer cell line (T3B12-5B) and **HeLa S3** cells support the use of these cells as substrates for the manufacture of a purified rAAV-HIV vaccine candidate. The data also supports the ability of the process, employing the **HeLa** cell substrate, used to manufacture the rAAV-HIV vaccine to produce a product as free of adventitious agents as current testing procedures can document. Safety of the rAAV-HIV vaccine is currently being assessed in a Phase I clin. trial.
REFERENCE COUNT: 31 THERE ARE 31 CITED REFERENCES AVAILABLE FOR THIS RECORD. ALL CITATIONS AVAILABLE IN THE RE FORMAT

L7 ANSWER 4 OF 15 CAPLUS COPYRIGHT 2006 ACS on STN
ACCESSION NUMBER: 2005:60754 CAPLUS
Correction of: 2004:1036571
DOCUMENT NUMBER: 142:233342
Correction of: 142:16836
TITLE: Sequences of human schizophrenia related genes and use for diagnosis, prognosis and therapy
INVENTOR(S): Liew, Choong-Chin
PATENT ASSIGNEE(S): Chondrogene Limited, Can.
SOURCE: U.S. Pat. Appl. Publ., 156 pp., Cont.-in-part of U.S. Ser. No. 802,875.
CODEN: USXXCO
DOCUMENT TYPE: Patent
LANGUAGE: English
FAMILY ACC. NUM. COUNT: 47
PATENT INFORMATION:

PATENT NO.	KIND	DATE	APPLICATION NO.		DATE
US 2004241727	A1	20041202	US 2004-812731		20040330
US 2004014059	A1	20040122	US 2002-268730		20021009
US 2005191637	A1	20050901	US 2004-803737		20040318
US 2005196762	A1	20050908	US 2004-803759		20040318
US 2005196763	A1	20050908	US 2004-803857		20040318
US 2005196764	A1	20050908	US 2004-803858		20040318
US 2005208505	A1	20050922	US 2004-803648		20040318
US 2004241727	A1	20041202	US 2004-812731		20040330
US 2004241727	A1	20041202	US 2004-812731		20040330
US 2004265869	A1	20041230	US 2004-812716		20040330
US 2005208519	A1	20050922	US 2004-989191		20041115
PRIORITY APPLN. INFO.:			US 1999-115125P	P	19990106
			US 2000-477148	B1	20000104
			US 2002-268730	A2	20021009
			US 2003-601518	A2	20030620
			US 2004-802875	A2	20040312
			US 2004-812731	A	20040330
			WO 2004-US20836	A2	20040621

AB The present invention is directed to detection and measurement of gene transcripts and their equivalent nucleic acid products in blood. Specifically provided is anal. performed on a drop of blood for detecting, diagnosing and monitoring diseases using gene-specific and/or tissue-specific primers. The present invention also describes methods by which delineation of the sequence and/or quantitation of the expression levels of disease-specific genes allows for an immediate and accurate diagnostic/prognostic test for disease or to assess the effect of a particular treatment regimen. [This abstract record is one of 3 records for this document necessitated by the large number of index entries required to fully index the document and publication system constraints.].

L7 ANSWER 5 OF 15 CAPLUS COPYRIGHT 2006 ACS on STN
ACCESSION NUMBER: 2004:899614 CAPLUS
TITLE: Method for production of oncolytic **adenoviruses**
INVENTOR(S): Kadan, Michael; Kaptur, Ronald; Brousseau, David; Mittelstaedt, Denise; Li, Yuanhao
PATENT ASSIGNEE(S): Novartis Ag, Switz.
SOURCE: PCT Int. Appl.
CODEN: PIXXD2
DOCUMENT TYPE: Patent
LANGUAGE: English
FAMILY ACC. NUM. COUNT: 1
PATENT INFORMATION:

PATENT NO.	KIND	DATE	APPLICATION NO.	DATE
WO 2004092348	A2	20041028	WO 2004-US11855	20040415
WO 2004092348	A3	20050310		
W: AE, AG, AL, AM, AT, AU, AZ, BA, BB, BG, BR, BW, BY, BZ, CA, CH, CN, CO, CR, CU, CZ, DE, DK, DM, DZ, EC, EE, EG, ES, FI, GB, GD, GE, GH, GM, HR, HU, ID, IL, IN, IS, JP, KE, KG, KP, KR, KZ, LC, LK, LR, LS, LT, LU, LV, MA, MD, MG, MK, MN, MW, MX, MZ, NA, NI, NO, NZ, OM, PG, PH, PL, PT, RO, RU, SC, SD, SE, SG, SK, SL, SY, TJ, TM, TN, TR, TT, TZ, UA, UG, US, UZ, VC, VN, YU, ZA, ZM, ZW				
RW: BW, GH, GM, KE, LS, MW, MZ, SD, SL, SZ, TZ, UG, ZM, ZW, AM, AZ, BY, KG, KZ, MD, RU, TJ, TM, AT, BE, BG, CH, CY, CZ, DE, DK, EE, ES, FI, FR, GB, GR, HU, IE, IT, LU, MC, NL, PL, PT, RO, SE, SI, SK, TR, BF, BJ, CF, CG, CI, CM, GA, GN, GQ, GW, ML, MR, NE, SN, TD, TG				
US 2005095705	A1	20050505	US 2004-824796	20040414
PRIORITY APPLN. INFO.:			US 2003-463143P	P 20030415

AB **HeLa-S3** cells comprising replication-competent **adenovirus** vectors are provided. Also provided are **HeLa -S3** producer cell lines and methods for producing replication-competent **adenovirus** using the same.

L7 ANSWER 6 OF 15 CAPLUS COPYRIGHT 2006 ACS on STN

ACCESSION NUMBER: 1998:268518 CAPLUS
DOCUMENT NUMBER: 128:318022
TITLE: DNA sequences encoding fusions of DNA repair proteins and their uses for inhibition action of chemotherapy agents in non-target cells
INVENTOR(S): Kelley, Mark; Williams, David
PATENT ASSIGNEE(S): Advanced Research & Technology Institute, USA
SOURCE: PCT Int. Appl., 150 pp.
CODEN: PIXXD2
DOCUMENT TYPE: Patent
LANGUAGE: English
FAMILY ACC. NUM. COUNT: 1
PATENT INFORMATION:

PATENT NO.	KIND	DATE	APPLICATION NO.	DATE
WO 9817684	A2	19980430	WO 1997-US19629	19971024
W: AL, AM, AT, AU, AZ, BA, BB, BG, BR, BY, CA, CH, CN, CU, CZ, DE, DK, EE, ES, FI, GB, GE, GH, HU, ID, IL, IS, JP, KE, KG, KP, KR, KZ, LC, LK, LR, LS, LT, LU, LV, MD, MG, MK, MN, MW, MX, NO, NZ, PL, PT, RO, RU, SD, SE, SG, SI, SK, SL, TJ, TM, TR, TT, UA, UG, US, UZ, VN, YU, ZW, AM, AZ, BY, KG, KZ, MD, RU, TJ, TM				
RW: GH, KE, LS, MW, SD, SZ, UG, ZW, AT, BE, CH, DE, DK, ES, FI, FR, GB, GR, IE, IT, LU, MC, NL, PT, SE, BF, BJ, CF, CG, CI, CM, GA, GN, ML, MR, NE, SN, TD, TG				
AU 9851953	A1	19980515	AU 1998-51953	19971024
US 6046036	A	20000404	US 1997-957302	19971024
US 6252048	B1	20010626	US 2000-542403	20000403
PRIORITY APPLN. INFO.:			US 1996-29308P P	19961025
			US 1997-957302 A1	19971024
			WO 1997-US19629 W	19971024

AB Described are DNA-repair fusion proteins of multiple DNA repair proteins possessing the activity of each protein, and their related polynucleotide sequences and vectors. Thus, chimeric proteins were generated from human O-6-methylguanine-DNA methyltransferase (MGMT) and human apurinic/apyrimidinic endonuclease (APE). The chimera included fusions of full-length proteins and with N-terminally truncated APE. Overlapping PCR techniques were used to construct expression vectors in which the cDNAs are expressed by the murine phosphoglycerate kinase promoter and containing the SV40 small T intron and poly(A) tract. The proteins, when expressed in cells, e.g., hematopoietic cells, increase the survival rate of the cells when contacted with chemotherapeutic agents. In mammalian **HeLa** cells, chimerics expressing MGMT-APE have a 2-fold survival enhancement over untreated **HeLa** cells at a BCNU [1,3-bis(2-chloroethyl)-1-nitrosourea] of 75 μM, whereas at 150 μM BCNU there is a 4-8 fold enhancement. Protection was also observed against MMS (Me methanesulfonate). Also described are transgenic animal models wherein these proteins are expressed in essentially all cells of the animal. Such animal models are useful for instance in testing chemotherapeutic agents.

L7 ANSWER 7 OF 15 CAPLUS COPYRIGHT 2006 ACS on STN
ACCESSION NUMBER: 1996:554297 CAPLUS
DOCUMENT NUMBER: 125:187476
TITLE: Selection of the best target site for ribozyme-mediated cleavage within a fusion gene for **adenovirus** E1A-associated 300 kDa protein (p300) and luciferase
AUTHOR(S): Kawasaki, Hiroaki; Ohkawa, Jun; Tanishige, Norie; Yoshinari, Koichi; Murata, Takehide; Yokoyama, Kazunari K.; Taira, Kazunari
CORPORATE SOURCE: National Inst. Biosci. Human Technology, Agency Industrial Sci. Technology, Tsukuba Science City, 305, Japan
SOURCE: Nucleic Acids Research (1996), 24(15), 3010-3016
CODEN: NARHAD; ISSN: 0305-1048
PUBLISHER: Oxford University Press
DOCUMENT TYPE: Journal
LANGUAGE: English

AB The cellular 300-kDa protein known as p300 is a target for the adenoviral E1A oncoprotein and it is thought to participate in prevention of the G0/G1 transition during the cell cycle, in activation of certain enhancers and in the stimulation of differentiation pathways. To determine the exact function of p300, as a first step we constructed a simple assay system for the selection of a potential target site of a hammerhead ribozyme in vivo. For the detection of ribozyme-mediated cleavage, we used a fusion gene (p300-luc) that considered of the sequence encoding the N-terminal region of p300 and the gene for luciferase, as the reporter gene. We were also interested in the correlation of the GUX rule, for the triplet adjacent to the cleavage site, with ribozyme activity in vivo. Therefore, we selected 5 target sites that all included GUX. The rank order of activities in vitro indeed followed the GUX rule; with respect to the kcat, a C residue as the third base (X) was the best, next came an A residue and a U residue was the worst (GUC > GUA > GUU). However, in vivo the tRNAVal promoter-driven ribozyme, targeted to a GUA located upstream of the initiation codon, had the highest inhibitory effect (96%) in **HeLa S3** cells when the molar ratio of the DNA template for the target p300 RNA to that for the ribozyme was 1:4. Since the rank order of activities in vivo did not conform to the GUX rule, it is unlikely that the rate limiting step for cleavage of the p300-luc mRNA was the chemical step. This kind of ribozyme expression system should be extremely useful for elucidation of the function of p300 in vivo.

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L7 ANSWER 8 OF 15 CAPLUS COPYRIGHT 2006 ACS on STN
ACCESSION NUMBER: 1994:676563 CAPLUS
DOCUMENT NUMBER: 121:276563
TITLE: Vitronectin receptor antibodies inhibit infection of **HeLa** and A549 cells by **adenovirus** type 12 but not by **adenovirus** type 2
AUTHOR(S): Bai, Mei; Campisi, Lauren; Freimuth, Paul
CORPORATE SOURCE: Biology Department, Brookhaven National Laboratory, Upton, NY, 11973, USA
SOURCE: Journal of Virology (1994), 68(9), 5925-32
CODEN: JOVIAM; ISSN: 0022-538X
DOCUMENT TYPE: Journal
LANGUAGE: English
AB The penton base gene from **adenovirus** type 12 (Ad12) was sequenced and encodes a 497-residue polypeptide, 74 residues shorter than the penton base from Ad2. The Ad2 and Ad12 proteins are highly conserved at the amino- and carboxy-terminal ends but diverge radically in the central region, where 63 residues are missing from the Ad12 sequence. Conserved within this variable region is the sequence Arg-Gly-Asp (RGD), which, in the Ad2 penton base, binds to integrins in the target cell membrane, enhancing the rate or the efficiency of infection. The Ad12 penton base was expressed in Escherichia coli, and the purified refolded protein assembled in vitro with Ad2 fibers. In contrast to the Ad2 penton base, the Ad12 protein failed to cause the rounding of adherent cells or to promote attachment of **HeLa S3** suspension cells; however, A549 cells did attach to surfaces coated with either protein and pretreatment of the cells with an integrin αvβ5 monoclonal antibody reduced attachment to background levels. Treatment of **HeLa** and A549 cells with integrin αvβ3 or αvβ5 monoclonal antibodies or with an RGD-containing fragment of the Ad2 penton base protein inhibited infection by Ad12 but had no effect on and in some cases enhanced infection by Ad2. Purified Ad2 fiber protein reduced the binding of radiolabeled Ad2 and Ad12 virions to **HeLa** and A549 cells nearly to background levels, but the concns. of fiber that strongly inhibited infection by Ad2 only weakly inhibited Ad12 infection. These data suggest that αv-containing integrins alone may be sufficient to support infection by Ad12 and that this pathway is not efficiently used by Ad2.

L7 ANSWER 9 OF 15 CAPLUS COPYRIGHT 2006 ACS on STN
ACCESSION NUMBER: 1994:316909 CAPLUS
DOCUMENT NUMBER: 120:316909

TITLE: Incorporation of **adenovirus** into a ligand-based DNA carrier system results in retention of original receptor specificity and enhances targeted gene expression
AUTHOR(S): Wu, George Y.; Zhan, Peili; Sze, Lillian L.; Rosenberg, Arielle R.; Wu, Catherine H.
CORPORATE SOURCE: Sch. Med., Univ. Connecticut, Farmington, CT, 06030, USA
SOURCE: Journal of Biological Chemistry (1994), 269(15), 11542-6
CODEN: JBCHA3; ISSN: 0021-9258
DOCUMENT TYPE: Journal
LANGUAGE: English

AB **Adenovirus** type 5 was modified by coupling an asialoglycoprotein-polylysine conjugate to the virus by reactions that activate carbohydrate residues. Wild-type virus modified in this manner had greatly decreased infectivity toward normally susceptible **HeLa S3** (asialoglycoprotein receptor (-)) and SK Hep1 (asialoglycoprotein receptor (-)) cells leaving 91 and 86% viable, resp., after 48 h. However, with Huh 7 (asialoglycoprotein receptor (+)) cells, modified virus retained its infectivity leaving only 19% of cells viable under identical conditions. Modified virus was complexed to DNA in the form of a plasmid, pSVHBV surf, containing the gene for hepatitis B surface antigen as a marker of gene expression. Huh 7, receptor (+), cells treated with modified wild type, and modified replication-defective dL312 virus complexed to DNA raised antigen levels by approx. 13- and 30-fold, resp., compared with asialoglycoproteinpolylysine DNA complex alone. Competition with a large excess of an asialoglycoprotein blocked the enhancement by more than 95%. Using a β-galactosidase marker gene, the number of cells transfected by modified virus was found to be 200-fold higher than complex alone. Yet, specificity was retained exclusively for asialoglycoprotein receptor-bearing cells. These data indicate that **adenovirus** can be chemical modified by coupling ligands resulting in targeted gene expression dictated specifically by receptor recognition of the attached ligand.

L7 ANSWER 10 OF 15 CAPLUS COPYRIGHT 2006 ACS on STN
ACCESSION NUMBER: 1985:466423 CAPLUS
DOCUMENT NUMBER: 103:66423
TITLE: Analysis of N-methyl-N'-nitro-N-nitrosoguanidine (MNNG)-induced DNA damage in tumor cell strains from Japanese patients and demonstration of MNNG hypersensitivity of Mer- xenografts in athymic nude mice
AUTHOR(S): Watatani, Masahiro; Ikenaga, Mituo; Hatanaka, Toshihiro; Kinuta, Masakatsu; Takai, Shinichiro; Mori, Takesada; Kondo, Sohei
CORPORATE SOURCE: Sch. Med., Osaka Univ., Osaka, 553, Japan
SOURCE: Carcinogenesis (1985), 6(4), 549-53
CODEN: CRNGDP; ISSN: 0143-3334
DOCUMENT TYPE: Journal
LANGUAGE: English

AB Among 15 human tumor cell strains from Japanese patients, 1 strain derived from a patient with thyroid cancer showed the inability to support the growth of **adenovirus** 5 treated with MNNG [70-25-7]. When plated on this Mer- strain, **adenovirus** 5 showed 3-4 times higher sensitivity to MNNG-induced killing than when plated on any of the other 14 Mer+ tumor cell strains. Biochem. anal. showed that the Mer- strain was defective in demethylation repair of O6-methylguanine [20535-83-5] produced by MNNG treatment. The sensitivities of 12 of 15 human tumor strains, including the Mer- strain, to MNNG were compared by measuring their colony-forming abilities. All the strains tested showed the Rem- phenotype (having higher sensitivity to MNNG-produced cell killing than normal fibroblasts). The differential killing effects of MNNG on Mer- and Mer+ tumor cells under in vivo conditions were tested using the Mer+ **HeLa S3** strain and its Mer- variant. Mer+ and Mer- cells were implanted s.c. into the left and right flanks, resp., of 10 nude mice and the next day, MNNG solution (0.25 mL at 1 mg/mL) was injected into the implantation site of 8 mice. Mer- tumor cells in 6 of 8 treated

mice showed no growth and those in the other 2 mice did grow, but regressed after .apprx.3 wk. In contrast, Mer+ tumor cells continued to grow in all the 8 mice treated, indicating that Mer- tumor cells may be selectively inactivated by suitable therapeutic regimens with appropriate methylating drugs.

L7 ANSWER 11 OF 15 CAPLUS COPYRIGHT 2006 ACS on STN
ACCESSION NUMBER: 1980:72508 CAPLUS
DOCUMENT NUMBER: 92:72508
TITLE: Kinetics of **adenovirus** DNA replication. I. Rate of **adenovirus** DNA replication
AUTHOR(S): Bodnar, John W.; Pearson, George D.
CORPORATE SOURCE: Dep. Biochem. Biophys., Oregon State Univ., Corvallis, OR, 97331, USA
SOURCE: Virology (1980), 100(1), 208-11
CODEN: VIRLAX; ISSN: 0042-6822
DOCUMENT TYPE: Journal
LANGUAGE: English
AB The rate of **adenovirus** DNA replication in **HeLa S3** cells was constant throughout infection. The average rate of replication was 0.046 fractional lengths/min or 1600 nucleotides/min. The time required to synthesize an **adenovirus** DNA mol. was 21.7 min.

L7 ANSWER 12 OF 15 CAPLUS COPYRIGHT 2006 ACS on STN
ACCESSION NUMBER: 1978:149950 CAPLUS
DOCUMENT NUMBER: 88:149950
TITLE: Involvement of microtubules in cytopathic effects of animal viruses: early proteins of **adenovirus** and herpesvirus inhibit formation of microtubular paracrystals in **HeLa-S3** cells
AUTHOR(S): Ebina, T.; Satake, M.; Ishida, N.
CORPORATE SOURCE: Dep. Bacteriol., Tohoku Univ. Sch. Med., Sendai, Japan
SOURCE: Journal of General Virology (1978), 38(3), 535-48
CODEN: JGVIAY; ISSN: 0022-1317
DOCUMENT TYPE: Journal
LANGUAGE: English
AB In order to exam. the involvement of microtubles in the virus-induced cytopathic effect (c.p.e.), the effect of virus infection on the formation of microtubular paracrystals (PC) induced by 10 μg/mL of vinblastine sulfate in **HeLa-S3** cells was examined by phase-contrast microscopy. In poliovirus-infected cells, c.p.e. (cell rounding) and the inhibition of PC formation proceeded in parallel, starting 4 h post-infection. In Sendai virus-infected cells, however, PC formation was not inhibited even 24 h postinfection, when most infected cells clearly showed c.p.e. (syncytial formation). In **adenovirus**-infected cells, the inhibition of PC formation was observed 9 h before the appearance of c.p.e. Cytosine arabinoside (ara C) did not block the inhibition of PC formation in infected cells, but blocked the appearance of late c.p.e. (nuclear alteration). Cycloheximide blocked both the inhibition of PC formation and the induction of late c.p.e. These results suggest that an early protein synthesized de novo by **adenovirus** is required for direct or indirect inhibition of the microtubular PC formation. Furthermore, on UV inactivation of **adenovirus** both activities (induction of early c.p.e. shown by shrinkage of cytoplasm, and inhibition of PC formation) followed the same inactivation curve and were inactivated at a slower rate than viral infectivity and the activity leading to late c.p.e. The UV light sensitive target responsible for the induction of early c.p.e. and the inhibition of PC formation was .apprx.20% of that for infectivity and was in accord with the genome size of the early functioning virus genes. In herpes simplex virus (HSV)-infected cells, the inhibition of PC formation, the appearance of c.p.e. (cell rounding and disappearance of nucleoli), and the synthesis of V antigen proceeded in parallel. These 3 functions of HSV were not blocked in infected cells even when the de novo synthesis of virus DNA was inhibited by ara C or phosphonoacetic acid (PAA), whereas these 3 functions were blocked by cycloheximide, suggesting that a protein coded by the input virus genome early after infection inhibits the microtubular PC formation and is responsible for c.p.e. From the UV inactivation curve of HSV, it was confirmed that only one-tenth of the virus genome was responsible for both

activities.

L7 ANSWER 13 OF 15 CAPLUS COPYRIGHT 2006 ACS on STN
ACCESSION NUMBER: 1978:133059 CAPLUS
DOCUMENT NUMBER: 88:133059
TITLE: Characterization of **adenovirus** RNA synthesized in the presence of an adenosine analog: failure of poly(A) addition
AUTHOR(S): Swart, C.; Hodge, L. D.
CORPORATE SOURCE: Dep. Hum. Genet., Yale Univ. Sch. Med., New Haven, CT, USA
SOURCE: Virology (1978), 84(2), 374-89
CODEN: VIRLAX; ISSN: 0042-6822
DOCUMENT TYPE: Journal
LANGUAGE: English
AB Synthesis of **adenovirus**-specific RNA in the presence of toyocamycin, an adenosine analog, late in infection of **HeLa S3** cells has been investigated. The effect of this analog on nuclear metabolism has been examined because, under the appropriate conditions, there is an apparent accumulation of rapidly sedimenting nuclear viral RNA (HnRNA) and no new viral mRNA assocs. with polyribosomes. Under these conditions there was an .apprx.10% substitution by toyocamycin for adenosine in viral HnRNA. A similar amount was incorporated into virus-associated RNA(s) but there was little effect on the synthesis, the size, or the appearance in the cytoplasm of this species of viral RNA. In the presence of the analog no polyadenylate-rich segments could be detected in nuclear viral RNA. Two 5' termini containing the methylated components 7-methyl-GMP and 6-methyl-AMP were recovered and constituted proportionately the same amount in selected RNA sequences whether or not synthesis had occurred in the presence of the adenosine analog. Relative to the recovery of 5' termini, selectively extracted RNA synthesized in the presence of toyocamycin yielded nearly 2-fold less 6-methyl-AMP. Since rapid sedimentation of nuclear viral RNA implies incomplete processing of mols., these results suggest that the incorporation of toyocamycin interferes with RNA metabolism because of its prevention of polyadenylation and(or) reduction in methylation of internal adenosine residues. The data also imply a sequence of events in which the introduction of at least some 5' alterations and internal methylations can occur prior to and independent of polyadenylate addns.

L7 ANSWER 14 OF 15 CAPLUS COPYRIGHT 2006 ACS on STN
ACCESSION NUMBER: 1977:28332 CAPLUS
DOCUMENT NUMBER: 86:28332
TITLE: Nuclear matrix of **HeLa S3** cells. Polypeptide composition during **adenovirus** infection and in phases of the cell cycle
AUTHOR(S): Hodge, L. D.; Mancini, P.; Davis, F. M.; Heywood, P.
CORPORATE SOURCE: Sch. Med., Yale Univ., New Haven, CT, USA
SOURCE: Journal of Cell Biology (1977), 72(1), 194-208
CODEN: JCLBA3; ISSN: 0021-9525
DOCUMENT TYPE: Journal
LANGUAGE: English
AB A subnuclear fraction has been isolated from **HeLa S3** nuclei after treatment with high salt buffer, deoxyribonuclease, and dithiothreitol. Ultrastructural and biochem. analyses indicated that this structure consisted of nonmembranous and membranous elements. Its chemical composition was 87% protein, 12% phospholipid, 1% DNA, and 0.1% RNA by weight The protein constituents were resolved in Na dodecyl sulfate polyacrylamide slab gels into 30-35 distinguishable bands in the apparent mol. weight range of 14,000-200,000 with major peptides at 14,000-18,000 and 45,000-75,000. Anal. of newly synthesized polypeptides by cylindrical gel electrophoresis revealed another cluster in the 90,000-130,000 mol. weight range. Infection with **adenovirus** resulted in an altered polypeptide profile. Addnl. polypeptides with apparent mol. wts. of 21,000, 23,000, and 92,000 became major components by 22 h after infection and some peptides in the 45,000-75,000 mol. weight range became less prominent. In synchronized cells the relative staining capacity of the 6 bands in the 45,000-75,000 mol. weight range changed during the cell cycle. Synthesis of at least some matrix polypeptides occurred in all phases of the cell cycle, although

there was decreased synthesis in late S/G2. In the absence of protein synthesis after cell division, at least some polypeptides in the 45,000-75,000 mol. weight range survive nuclear dispersal and subsequent reformation during mitosis.

L7 ANSWER 15 OF 15 CAPLUS COPYRIGHT 2006 ACS on STN
ACCESSION NUMBER: 1968:504862 CAPLUS
DOCUMENT NUMBER: 69:104862
TITLE: Studies on photosensitizing dyes. III. Effect of photosensitizing dyes on infected cells
AUTHOR(S): Ogasawara, Hisayasu
CORPORATE SOURCE: Med. Sch., Okayama Univ., Okayama, Japan
SOURCE: Kanko Shikiso (1968), No. 73, 17-20
CODEN: KASHAJ; ISSN: 0461-5956
DOCUMENT TYPE: Journal
LANGUAGE: Japanese
AB The inhibitory effect of the photosensitizing dyes of cyanine and aminovinyl compds. on the growth of **HeLa-S3** cells, in tissue culture infected with **adenovirus** type 12 or with measles virus, was studied. The cyanine compds., such as 4,4-dimethyl-3,3'-di-n-heptyl-2,2'-thiazolocyanine nicotinate, 1,1-diethyl-11-[4-(1-ethyl quinoline)]-4,4'-dicarbocyanine di-L-aspartate, and 1,1'-diethyl-11-[4-(1-ethyl quinoline)]-4,4'-dicarbocyanine diglucuronate, 6-methyl-1-isopropyl-2-[2-(5-bromo-2-pyridylamino)vinyl]-pyridinium iodide, 1-ethyl-6-methyl-2-[2-(5-iodo pyrimidylamino)vinyl]-pyridinium iodide, 3,4-dimethyl-2-(2-pyrimidyl-aminovinyl) oxazolium iodide, and 3,4-dimethyl-2-(2-anilino vinyl) oxazolium iodide, showed a slight inhibitory effect on the proliferation of **HeLa-S3** cells infected with **adenovirus** type 12, while that of cells infected with measles virus was not affected by these photosensitizing dyes.

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